L3 ANSWER 251 OF 266 CAPLUS COPYRIGHT 1999 ACS DUPLICATE 102
AN 1989:93290 CAPLUS
DN 110:93290
TI Effect of **interferons** and poly(I):poly(C) on the pathogenesis of the diabetogenic variant of encephalomyocarditis virus in different mouse strains
AU Giron, David J.; Agostini, Heidi J.; Thomas, Donald C.
CS Coll. Sci. Math., Wright State Univ., Dayton, OH, USA
SO J. Interferon Res. (1988), 8(6), 745-53
CODEN: JIREDJ; ISSN: 0197-8357
DT Journal
LA English
CC 15-5 (Immunochemistry)
Section cross-reference(s): 1
AB **Interferon** (IFN) can either **prevent** or exacerbate the pathogenic effects of the diabetogenic variant of encephalomyocarditis (EMC-D) virus. The effect seen is dependent upon the mouse strain and the time of IFN administration. Studies were initiated to investigate the role of the IFN system in the pathogenesis of this virus infection. Here IFNs or poly(I):poly(C) were administered to several mouse strains at 24 h before or 4 days after infection with EMC-D virus. The results of such treatment ranged from complete protection of the animals from the diabetogenic effects of the virus to exacerbation of the infection as reflected by the virus content in selected organs. The effect was dependent upon the mouse strain, the type of IFN, and the time of its administration in relation to virus infection.
ST **interferon diabetes** encephalomyocarditis virus infection pathogenesis; polyinosinate polycytidylate **diabetes** encephalomyocarditis pathogenesis
IT Mouse
(**diabetes** induced in, by encephalomyocarditis virus, **interferon** and **interferon** inducer effect on, strain-dependent)
IT **Diabetes** mellitus
(encephalomyocarditis virus-induced, pathogenesis of, **interferon** and **interferon** inducer effect on, factors modulating)
IT Genetics
(of **interferon** and **interferon** inducer effect on pathogenesis of encephalomyocarditis virus-induced **diabetes**, in mouse strains)
IT Virus, animal
(encephalomyocarditis, **diabetes** induced by, pathogenesis of, **interferon** and **interferon** inducer effect on, factors modulating)
IT **Interferons**
RL: BIOL (Biological study)
(.alpha./.beta., encephalomyocarditis virus-induced **diabetes** pathogenesis response to, factors modulating)
IT 24939-03-5, Poly(I):poly(C)
RL: BIOL (Biological study)
(encephalomyocarditis virus-induced **diabetes** pathogenesis response to, factors modulating)

.16 ANSWER 89 OF 101 CA COPYRIGHT 1995 ACS DUPLICATE 42
.N 108:4454 CA
'I Toxicity studies of human fibroblast interferon beta (I). Acute and subacute toxicity studies in mice and rats
.U Shibutani, Yasunori; Obata, Masaomi; Hamada, Yoshimasa; Shichi, Shigeo; Ohi, Keiichi; Kaga, Nobuhiko; Yajima, Gompachi
:S Toxicol. Lab., Mochida Pharm. Co., Ltd., Gotemba, 412, Japan
:O Iyakuhin Kenkyu (1987), 18(4), 571-82
CODEN: IYKEDH
)T Journal
.A Japanese
.B In an acute toxicity study, i.v. or oral administration of 1 .times. 107 - 2.5 .times. 108 IU and i.m. administration of 1 .times. 107 - 5 .times. 107 IU of human interferon .beta. (MR 21)/kg caused no death, apparent symptoms, body wt. change or abnormal autopsy findings in mice and rats. I.v., i.m., and oral LD50 values of MR-21 in mice and rats were >2.5 .times. 108, >5 .times. 107, and >2.5 .times. 108 IU/kg, resp. In a subacute toxicity study, MR-21 administered i.v. to rats for 13 wk at 1 .times. 107 - 3 .times. 105 IU/kg/day caused no death or any symptoms attributable to the administration of MR-21. The no-effect dose level of MR-21 was estd. to be 3 .times. 105 IU/kg/day under the conditions of this study.

L6 ANSWER 1136 OF 1213 MEDLINE DUPLICATE 448
AN 86300315 MEDLINE
DN 86300315
TI [Alpha **interferon** in condylomata acuminata and juvenile **diabetes** mellitus].
Interferon-alpha bei Condylomata acuminata und juvenilem **Diabetes** mellitus.
AU Gross G; Roussaki A; Ikenberg H; Drees N
SO DEUTSCHE MEDIZINISCHE WOCHENSCHRIFT, (1986 Sep 5) 111 (36) 1351-5.
Journal code: ECL. ISSN: 0012-0472.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals; Cancer Journals
EM 198612
AB Persistent condylomata acuminata in a 21-year-old patient with **diabetes** mellitus were treated with highly purified **interferon**-alpha (IFN-alpha) obtained by recombinant DNA technology. Daily dose was 1.5 X 10(6) IU, given subcutaneously. Already during treatment the condylomata regressed. Two weeks after the end of therapy, i.e. after a total dose of 10.5 X 10(6) IU IFN-alpha, all condylomata had completely receded. Blood glucose levels remained constant with concomitant insulin therapy. Toxic side-effects or antibodies to IFN-alpha were not observed.
CT Check Tags: Case Report; Comparative Study; Human; Male
Adult
Biopsy
Condylomata Acuminata: MI, microbiology
Condylomata Acuminata: PA, pathology
*Condylomata Acuminata: TH, therapy
Diabetes Mellitus, Insulin-Dependent: MI, microbiology
Diabetes Mellitus, Insulin-Dependent: PA, pathology
***Diabetes Mellitus, Insulin-Dependent: TH, therapy**
English Abstract
Interferon Type I: AE, adverse effects
***Interferon Type I: TU, therapeutic use**
Penile Neoplasms: MI, microbiology
Penile Neoplasms: PA, pathology
*Penile Neoplasms: TH, therapy
Penis: MI, microbiology
Penis: PA, pathology
Recombinant Proteins: AE, adverse effects
*Recombinant Proteins: TU, therapeutic use
CN 0 (**Interferon** Type I); 0 (Recombinant Proteins)

TI Antibodies to . ***alpha*** .- ***interferon*** in a patient with systemic lupus erythematosus.
AU Panem S.; Check I.J.; Henriksen D.; Vilcek J.
CS Dept. Pathol., Pritzker Sch. Med., Univ. Chicago, Chicago, IL 60637, United States
SO J. IMMUNOL., (1982) 129/1 (1-3).
CODEN: JOIMA3
CY United States
LA English
AB IFN is normally not demonstrable in the serum and other body fluids in the absence of an inducing stimulus, such as virus infection; however, IFN was found at high frequency in the sera of patients with autoimmune diseases including systemic lupus erythromatosus (SLE), ***rheumatoid*** ***arthritis*** , and Sjogren's syndrome. In this report we describe the identification of antibodies to IFN-.alpha. (leukocyte IFN) present at a very high titer in the serum of an SLE patient.

L71 ANSWER 512 OF 524 COPYRIGHT 1995 DERWENT INFORMATION LTD
AN 94-302673 [37] WPIDS
DNC C94-159283
TI Use of alpha- or ***beta*** - ***interferon*** or analyogues - for preventing or treating an autoimmune disorder, e.g. diabetes, arthritis, or transplant rejection.
DC B04 D16
IN SOBEL, D O
PA (GEOU) UNIV GEORGETOWN
CYC 18
PI WO 9420122 A1 940915 (9437)* 36 pp
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE
W: AU CA
AU 9463549 A 940926 (9503)
ADT WO 9420122 A1 WO 94-US2154 940307; AU 9463549 A AU 94-63549 940307
FDT AU 9463549 A Based on WO 9420122
PRAI US 93-26758 930305
AB WO 9420122 A UPAB: 941223
A method of preventing or treating an autoimmune disease in a mammal comprises administering at least one subtype of alpha- or ***beta*** - ***interferon*** or a hybrid or analogue of either or a mixt. Also claimed are:
(1) a method treating an asymptomatic preclinical autoimmune state in a mammal, which comprises administering a single subtype of alpha- or ***beta*** - ***interferon*** or a hybrid or analogue of either or a mixt.; (1) a method inhibiting rejection of transplanted islet cells or a pancreas in a mammal having transplanted islet cells or pancreas, comprising administering a single subtype of alpha- or ***beta*** - ***interferon*** or a hybrid or analogue or a mixt.
USE - The method can be used for treating or preventing autoimmune disorders such as type I ***diabetes*** ***mellitus*** , ***rheumatoid*** ***arthritis*** , systemic lupus erythematosus, scleroderma, sjogrens syndrome, mixed connective tissue disease, ankylosis spondylitis, Reiter's syndrome, psoriatic arthritis, hypersensitivity vasculitis, ulcerative colitis, cirrhosis, autoimmune uveitis, myasthenia gravis, Buerger's disease, Kawasaki's disease, systemic necrotising vasculitis, regional enteritis and hypoparathyroidism.
The interferon can be administered at a dose of e.g. 1x105 units to 75x106 units, e.g. orally.
Dwg.0/2

L71 ANSWER 513 OF 524 COPYRIGHT 1995 DERWENT INFORMATION LTD
AN 93-336896 [42] WPIDS
CR 93-336897 [42]

.16 ANSWER 89 OF 101 CA COPYRIGHT 1995 ACS DUPLICATE 42
.N 108:4454 CA
'I Toxicity studies of human fibroblast interferon beta (I). Acute and subacute toxicity studies in mice and rats
.U Shibutani, Yasunori; Obata, Masaomi; Hamada, Yoshimasa; Shichi, Shigeo; Ohi, Keiichi; Kaga, Nobuhiko; Yajima, Gompachi
:S Toxicol. Lab., Mochida Pharm. Co., Ltd., Gotemba, 412, Japan
:O Iyakuhin Kenkyu (1987), 18(4), 571-82
CODEN: IYKEDH
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.A Japanese
.B In an acute toxicity study, i.v. or oral administration of 1 .times. 107 - 2.5 .times. 108 IU and i.m. administration of 1 .times. 107 - 5 .times. 107 IU of human interferon .beta. (MR 21)/kg caused no death, apparent symptoms, body wt. change or abnormal autopsy findings in mice and rats. I.v., i.m., and oral LD50 values of MR-21 in mice and rats were >2.5 .times. 108, >5 .times. 107, and >2.5 .times. 108 IU/kg, resp. In a subacute toxicity study, MR-21 administered i.v. to rats for 13 wk at 1 .times. 107 - 3 .times. 105 IU/kg/day caused no death or any symptoms attributable to the administration of MR-21. The no-effect dose level of MR-21 was estd. to be 3 .times. 105 IU/kg/day under the conditions of this study.